33 Effects of pre- and/postnatal treatment with nicotine or postnatal exposure to cigarette smoke on hypothalamic catecholamine nerve terminal systems and neuroendocrine function in adult male and female rats.

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1. Male or female Sprague-Dawley rats were treated with nicotine during the prenatal and the first three postnatal weeks (the pregnant and lactating rats were given nicotine (+) tartrate (165mg/kg) in the tap water). Following one week of withdrawal (age 4 weeks) from pre- and postnatal treatment with nicotine an increased catecholamine (CA) utilization was observed in the medial (MPZ) and lateral (LPZ) palisade zones of the median eminence (ME) of both the male and female rats. This change maintained also at an age of 6-7 months, most prominent in the female diestrous rat and to a lesser degree in the male rat. In the adult male rat also increased noradrenaline (NA) stores were found in the paraventricular hypothalamic nucleus (PA). In the 4 week female rat reduced serum prolactin levels were found following pre- and postnatal nicotine treatment, an effect which was normalized in the adult rat. In the male rat no changes in serum TSH, prolactin, LH and corticosterone levels could be observed at an age of 4 weeks, but increased LH serum levels were found in the 6 month old rat.
2. Male rats were exposed to the smoke from 2 cigarettes (Kentucky reference IR-1 type) every morning from the day 1 after birth for a period of 5, 10 or 20 days. The rats were decapitated 24 h, 1 week or 7 months after last exposure. Serum LH levels were increased 24 h after a 10 and 20 day exposure to cigarette smoke . In adult life (6 months of age) following a 20 day postnatal exposure to cigarette smoke increased serum levels of corticosterone and prolactin were observed. Postnatal exposure to cigarette smoke produced increased CA utilization in the MPZ of the ME as well as a reduction in CA utilization in the PA 24 h after a 20 day exposure to cigarette smoke. These changes were not present in adult life.
The present results demonstrate that pre- and postnatal treatment with nicotine produces permanent activation CA nerve terminal systems of the external layer of the ME, mainly in the female rat.
Postnatal exposure to cigarette smoke produces hypersecretion of prolactin and corticosterone in the adult male rat in which hypothalamic CA nerve terminal systems seem not to play a major mediating role.
Taken together pre- and postnatal treatment with nicotine and postnatal exposure to cigarette smoke seem to produce functional consequences for neuroendocrine regulation as well as sex-specific responses in CA nerve terminal systems involved in food and water intake and in the response to stress in the male versus the female rat. A nicotine-induced disturbance in brain and pituitary cell replication and differantiation may underlie the permanent alterations observed.

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